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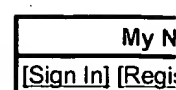
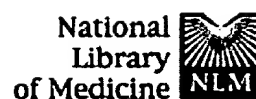
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could you please get the following ref(s):

Int J Cancer. 1996 Jan 17;65(2):204-8.

Cancer Immunol Immunother. 1995 Oct;41(4):236-42.

Christopher Yaen
US Patent Office
Art Unit 1643
571-272-0838
REM 3A20
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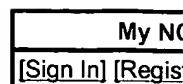
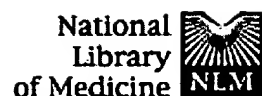
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Tumor-cell vaccination induces tumor dormancy in a murine model of B-cell leukemia/lymphoma (BCL1).

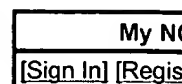
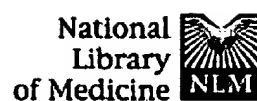
Morecki S, Pugatsch T, Levi S, Moshel Y, Slavin S.

Department of Bone-Marrow Transplantation, Hadassah University Hospital, Jerusalem, Israel.

Immunity to murine B-cell leukemia/lymphoma (BCL1) induced by multiple injections with irradiated tumor cells, prevented leukemia development in primary and adoptive transfer recipients despite long-lasting persistence of residual tumor cells. Detection of dormant BCL1 cells was carried out by PCR analysis using the VH-rearranged DNA sequence as a BCL1 clonal marker. Dormant tumor cells were detected > 250 days following immunity induction in 40% of spleens from healthy immune mice having no detectable symptoms of disease. Tumor dormancy was not abrogated by adoptive transfer of BCL1-containing splenocytes into syngeneic recipients, indicating that cell-mediated anti-tumor immunity contributes to maintenance of the tumor dormant state and prevents renewed tumor-cell growth. Splenocytes but not sera from immune mice conferred specific radiosensitive protection from a lethal dose of BCL1 cells included in cell mixtures transferred to secondary recipients. A therapeutic effect of transferred immune splenocytes was shown in BCL1-bearing mice, which remained disease-free for > 200 days after inoculation; nevertheless, dormant BCL1 cells were detected by PCR analysis in some of the surviving mice. Our results suggest that an efficient tumor-cell vaccine can lead to induction of tumor dormancy that can be maintained by a cell-derived mechanism for a long period of time.

PMID: 8567118 [PubMed - indexed for MEDLINE]

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Induction of tumor immunity by intact irradiated leukemic B cells (BCL1) bearing a tumor-associated cell-surface idiotype and the costimulatory B7 molecule.

Morecki S, Levi S, Puyesky Y, Slavin S.

Department of Bone Marrow Transplantation, Hadassah University Hospital, Jerusalem, Israel.

The idiotypic (Id) determinant of immunoglobulin expressed on the cell surface of malignant B cells represents a prototypical tumor-associated antigen (TAA), which has been used in a purified soluble form for active immunization in experimental tumor models and human hematological malignancies. Using a spontaneous transplantable murine model of B cell leukemia/lymphoma (BCL1), we have demonstrated the expression of the B7 costimulatory molecules in addition to the previously described Id determinant and class II major histocompatibility antigens. Intact irradiated BCL1 cells bearing these distinct determinants induced long lasting antitumor immunity in naive syngeneic mice. Induction was dose-dependent and most effective when three doses of 30×10^6 intact irradiated BCL1 cells were given at intervals of 7-10 days. The induced immunity protected 96% of 28 mice inoculated with a lethal dose of 10^5 - 10^6 nonirradiated BCL1 cells and 85% of 27 mice given a second challenge, whereas control mice died on day 20 after inoculation with 10^6 BCL1 cells. Adoptive transfer of splenocytes derived from immune mice did not induce leukemia in syngeneic recipients. Such splenocytes, harvested more than 365 days following immunization and administered together with fresh BCL1 cells to adoptive recipients, were able to confer protection for 90 days, even following a second challenge given 104 days after the first one. BCL1 immune splenocytes transferred into BCL1-bearing mice exerted a therapeutic effect, preventing leukemia onset for at least 180 days. Our results demonstrate the ability of tumor cells to trigger effective anti-tumor immunity. These findings could ultimately be applied to the prevention of tumor relapse in treatment of hematological and other malignancies expressing TAA, class II MHC antigen and costimulatory molecules.